1. Rice

(*Oryza sativa* L., Family: Poaceae)

1. Blast:

Rice blast disease is one of the most serious diseases of the rice plant worldwide. The disease was first recorded in 1637 in China. In India, the disease was first reported from Tamil Nadu in 1918 and later in other parts of the country. The disease is now reported in almost all rice producing states. The disease is caused severe yield loss ranging up to 80 per cent. Losses vary with stage of crop at infection. In India, more than 266,000 tons of rice were lost, which was about 0.8% of their total yield. A 10% neck rot causes yield loss of 6 % and 5% increases in chalky kernels.

Symptoms: The fungus is able to infect and produce lesions on all organs of the rice plant at all stages of life cycle except the root.

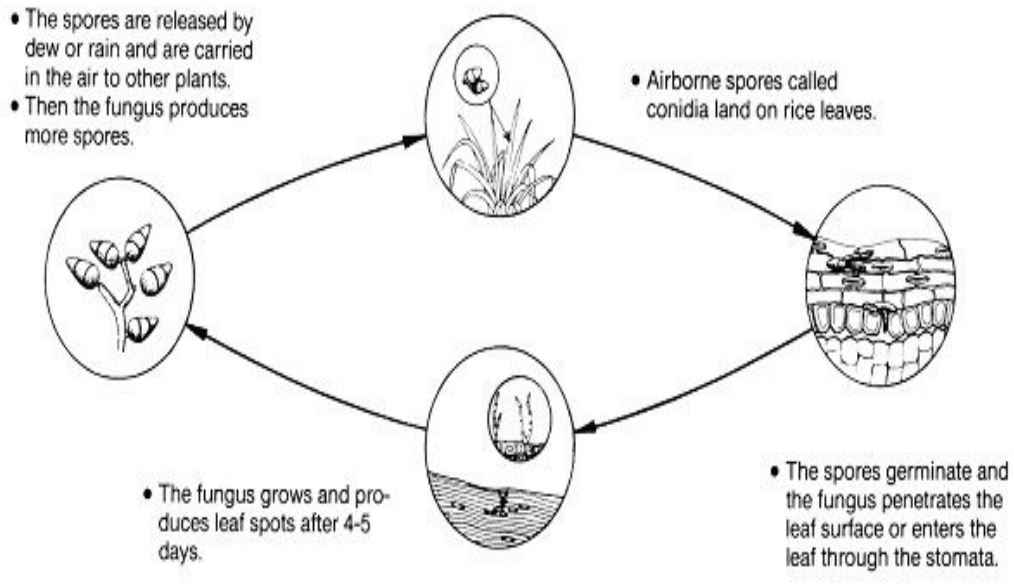
- **Leaf blast:** On the leaves, the lesions start as small water soaked bluish green specks, soon enlarge and form characteristic **spindle shaped** spots with grey centre and dark brown margin. The spots join together as the disease progresses and large areas of the leaves dry up and wither. Similar spots are also formed on the sheath. Severely infected nursery and field show a **burnt** appearance.
- **Node blast:** In infected nodes, irregular black areas that encircle the nodes can be noticed. The affected nodes may break up and all the plant parts above the infected nodes may die **(Node blast).**
- **Neck blast:** At the flower emergence, the fungus attacks the peduncle which is engirdled, and the lesion turns to brownish-black. This stage of infection is commonly referred to as rotten neck/neck rot/neck blast/panicle blast. In early neck infection, grain filling does not occur and the panicle remains erect like a **dead heart caused by a stem borer.**
- **Glume Blast:** In the late infection, partial grain filling occurs. Small brown to black spots also may be observed on glumes of the heavily infected panicles.

Causal organism: Rice blast is caused by the Ascomycete fungus, (Perfect/ telomorph: *Magnaporthe grisea* (Herbert) Yaegashi and Uddagawa (anamorph: *Pyricularia grisea* (Cooke) Sacc., synonym: *P. oryzae* Cav.). **The fungus produces septate, branched and**

hyaline to olivaceous mycelium. The conidiophores emerge out from leaf cuticle or through stomata that are simple, 2-4 septate and olivaceous in colour**. Conidia are hyaline to pale olive, pyriform to obclavate, 1-3 septate with a small basal appendage and 7-9 conidia are borne terminally on conidiophores.** The conidia are measure 20-25 x 8.5-9.5 µm. These germinate and develop an appressorium at the tip of the germ tube, which attaches to the surface of plant tissues; an infection-peg from the appresorium penetrates into plant tissues. The wall of conidiophores and appressorium are pigmented by melanin. **Sexual reproduction by formation of curved to fusiform four celled hyaline ascospores produced in perithecia.** A total of 32 physiological races of the pathogen were identified from different regions of the India which is based on the infection types developing on different hosts.

Disease Cycle:

- Primary infection by conidia from infected seed and plant debris.
- The disease perpetuates through diseased plant debris lying in the field, seed and wild grasses.
- The blast spore infects and produces a lesion on the rice plant and ends when the fungus sporulates repeatedly for about 20 days and disperses many new airborne spores.
- Under favourable moisture and temperature conditions (long periods of plant surface wetness, high humidity (93-99%), little or no wind at night and night temperatures between 12– 32 °C) the infection cycle can continue.
- In the canopy of rice plants, newly developed leaves act as receptors for the spores. The maximum number of spores produced was 20,000 on one lesion on leaves and 60,000 on one spikelet in one night.
- Lesions on leaves become an inoculum source for panicles. The pathogen can continue to live in plants from one crop season to another on residues of diseased plants or seeds, or on rations of stubble.
- Weeds can act as alternative/ collateral hosts (*Panicum repens, Digitaria marginata, Leerisa hexandra* etc.) for the disease development.
- Secondary infection through air borne conidia.



Disease Management:

- Diseased straw and stubble must be burned or composted otherwise they can become inoculum sources for the next crop season.
- Remove and destroy the weed hosts in the field bunds and channels.
- To obtain healthy seeds, the seeds must be collected from the field located under unfavorable conditions for the pathogen and fungicide must be applied if necessary.
- Nitrogen and phosphorus content in the plants affects disease proneness. Excess nitrogen fertilizer encourages disease development, while silica application reduces disease development. Split application of nitrogen and judicious application of nitrogenous fertilizers.
- Sowing into water eliminates disease transmission from seeds to seedlings because of the anaerobic condition that is unfavorable to the pathogen. On the contrary, sowing on wet soil allows seed transmission. Shade affects disease occurrence because of the longer wet condition.
- Some varieties are highly resistant and recommended for the cultivation in various agro-climatic zones of India such as IET, 1444, RPC 2B 849, CR 12-178 and Jaya (North India), T-603, T-141 (Orissa), A-67, A-90, A-200, A-249 (Maharashtra), GS- 397, GS-480, ADT- 20, CH- 20, Suchi, Alkulu, Gennibera, Kamala (Bihar), Co-4, TKM-1 (Tamil Nadu), IR- 579, Bala Late-6 (Himachal Pradesh), Jaya, Pankaj, Jayanti (Raipur, Madhya Pradesh). Simhapuri, Tikkana, Sriranga, Phalguna, Swarnadhan, Swarnamukhi, MTU 7414, MTU 9992, MTU 1005, Swathi, IR 64, IR 36, Sravani, Jaya, Vijaya, Ratna, RP 4-14, IET 1444, IR20, TKM 6, MTU-3 & 5 and NLR 9672 & 9674 in different tracts of Andhra Pradesh.
- Bhalum1, Bhalum2 for **upland** and Shasarang and Lampnah for transplanted **lowland**

- Seed treatment for removal of sterile, light seeds treatment with 3 % brine water followed by washing healthy seed with clean water.
- Seed treatment with Captan or Thiram or Carbendazim or Carboxin or Tricyclazole at 2 g/kg seed or biocontrol agent *Trichoderma viride*@ 4g/kg or *Pseudomonas fluorescens* @ 10g/kg of seed.
- Avoid close spacing of seedlings in the main field.
- Foliar spray of any one of fungicides viz., Edifenphos@0.1% or Carbendazim@0.1% or **Tricyclazole** @0.1% or Thiophanate Methyl@0.1% or copper oxychloride 0.3% or blasticidin 100 ppm or kasugamycin100 ppm (atibiotics) 2-3 sprays at an interval of 15 days as per severity of the disease.

2. Brown leaf Spot: (Brown spot / Sesame leaf spot / Helminthosporiose disease)

First report in India by Sundararaman–(1919) from Tamil Nadu. Rice brown spot is a historical disease because it caused a major outbreak in Bengal in India in 1942 when approximately two million people died of starvation. The disease is widely distributed in India, especially in West Bengal, Orissa, Andhra Pradesh and Tamil NaduIt was used by the USA as a biological weapon in Japan duringWorld War II.

Symptoms:

- In early stage it cause seedling blight. Leaf spot like sesamum seed size, at maturity coalesce to form large patches. Brown spots on glumes
- The symptoms first appear as brownish spot on leaves and glumes of the plant. The disease causes blight of seedlings.
- On seedlings, the pathogen produces small, brown lesions, which may girdle the coleoptile and cause distortion of the primary and secondary leaves.
- In some cases, pathogen may also infect and cause a black discolouration of rootsatt. Infected seedlings are stunted or killed.
- On the leaves of older plants, the pathogen produces circular to oval lesions that are grey at the centre and brown at the borders measuring about 0.5-2mm X 2-5mm.
- The fungus may also infect the glumes,causing dark brown to black oval spots, and may also infect the grain, causing a black discoloration

- On moderately susceptible cultivars, the pathogen produces tiny, dark specks. When infection is severe, the lesions may coalesce, killing large areas of affected leaves.
- The pathogen causes main damage by attacking the leaves at seedling stage.
- At times the neck region may be infected causing similar symptoms to those of neck blat caused by *P. oryzae*.
- The affected plants become weak and the yield is drastically reduced. The grains show a black discoloration.

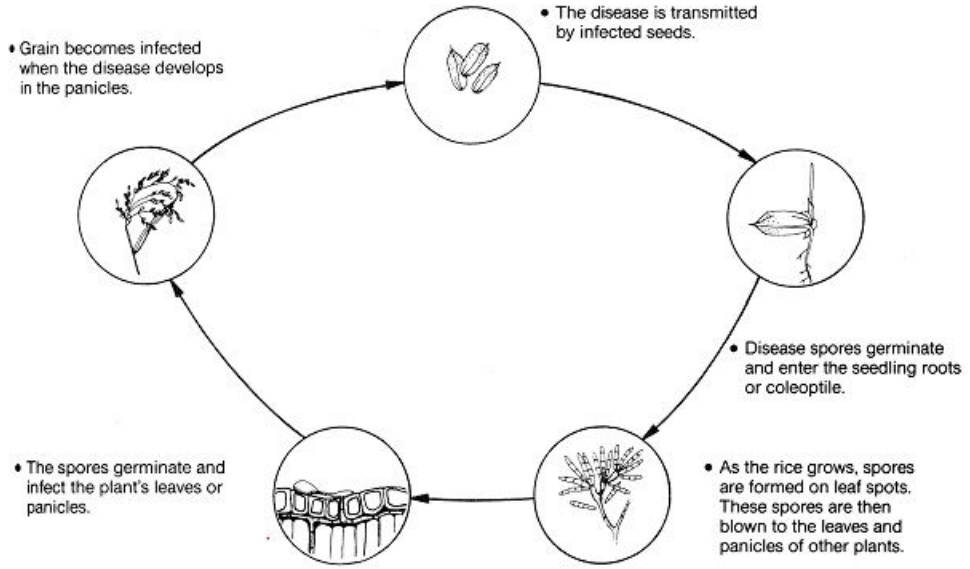

Pathogen: *Helminthosporium oryzae* Breda [Syn: *Bipolaris oryzae* Breda de Hann) Shoemaker*]*

- The perfect stage is **Cochliobolus miyabeanus** I to and Kubribayashi. *C. miyabeanus* belongs to the subdivision Ascomycotina, the sac fungi. The brown mycelium is inter or intracellular in the host. The conidiophores are multiseptate upto 600µm long and 4-8 µm wide and develop singly or in bundles (generally 17). Conidia are generally curved, boat or club shaped with 6-14 septa, 63-153 x 14-22 µm and often with a minute, slightly protruding hylum, by which they are attached to conidiophores. The conidia are born singly and successively at regular intervals on the upper part of the conidiophore in a sympodial manner.
- The conidia are dispersed by the wind. These spores are asexual; they do not rise from sexual crosses, but rather act as a method of dispersing the pathogen. A consequence of this is that the pathogen can spread rapidly in devastating epidemics. The conidia germinate readily, producing germ tubes mostly from both the end cells.
- The perithecia of the perfect stage pathogen (*C. miyabeanus*) are globose with the outer wall dark yellowish brown and parenchymatous, and with ostiolar beak.The asci are cylindrical, slightly curved and contain 4-6 ascospores.
- The ascospores are hyaline, long cylindrical with 6-15 septa and measuring 6-9 X 240-268 micron.

Disease Cycle: The fungus overwinters mainly in **infected plant parts**. The pathogen is not soil borne. This disease occurs naturally on as many as 20 different wild species of Oryzae. A few collateral hosts like *Digitaria sanguinalis, Echinochloa colona, Pennisetum typhoides*

Setaria italica and Cynodon dactylon, on which the pathogen is recorded, may serve as a source of primary inoculum.

- The conidia carried through infected rice seeds are also the **primary source** of inoculum. The conidia germinate on the emerging seedlings and produce several generations asexually as the plants grow.
- They are blown away by wind and cause infection to the new crop and thus serve as the source of secondary infection. The optimum temperature for the germination of conidia is 25-30C and humidity 90% or above. If seedlings are affected crop yield is drastically reduced. The pathogen can spread fast and may cause devastating epidemics.



Disease Management:

- Use disease free seed
- Avoid excesses of N –application
- Seed treatment with captan, thiram or carbendazim @ 0.2%
- Use of silicon fertilizers (e.g., calcium silicate slag)
- As the disease incites discolouration of the tissues, phenolic prohibitins present in rice tissues are associated with disease resistance.
- The reducing agents such as ascorbic acid and glutathione enhance the tissue susceptibility.
- The pathogen produces a toxin, obliobolin.
- Use of resistant varieties like BAM-10, CO-20, and IR-36 etc. are some of the resistant varieties.
- Since the fungus is seed transmitted, a hot water seed treatment (53-54°C) for 10-12 minutes and controls primary infection at the seedling stage.
- Plants growing in good nutritional conditions are generally resistant to the disease.
- The susceptibility to infection appears to be a deficiency in available silicon.
- Preventive treatment of the field with calcium silicate may be used in deficient soil.

- Seed treatment with tricyclazole followed by spraying of mancozeb + tricyclazole at tillering and late booting stages gave good control of the disease.
- Foliar spray of any one of fungicides viz., Edifenphos@0.1% or Carbendazim@0.1% or **Tricyclazole** @0.1%.

3. Bacterial Leaf Blight (BLB)

Economic Importance: It is also known as **poor man's disease/ dangereous/ killer disease**. Bacterial blight is reported to have reduced Asia's annual rice production by as much as 60%. BLB of rice had been known in Japan as endemic disease since 1881. About 300,000 to 400,000 hectares of rice were affected by the disease in recent years. There were 20% to 50% yield losses reported in severely infected fields. In Indonesia, losses were higher than those reported in Japan. **In India**, the bacterial blight was first recorded in 1951 in the Khopali area erstwhile **Bombay** state but at the time the pathogen was not identified (Bhapkar et. al. 1996). **The pathogen strain (*Xanthomonas oryzae*) of BLB was first time reported by Shriniwasan et. al (1959).** Due to this disease millions of hectares were severely infected, causing yield losses from 6% to 60%. With the introduction of rice variety Taichung Native-1 (TN-1 is highly susceptible to rice blast) in India the disease spread rapidly throughout the country.

Symptoms:

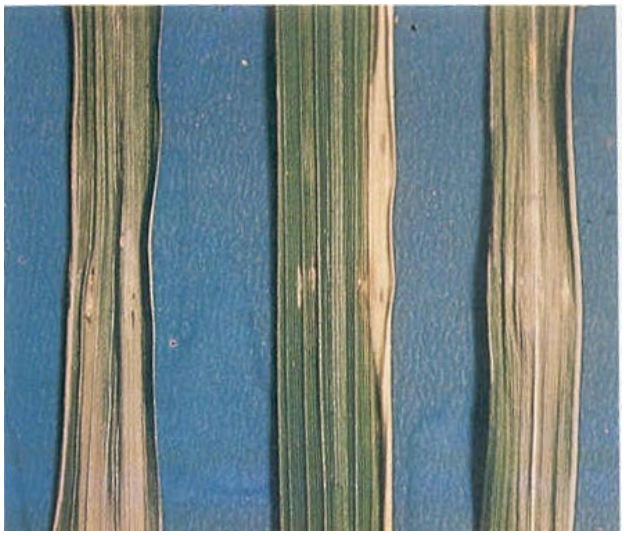
Kresek Phage/Wilt Phage

- Results from early systemic infection.
- Initial disease symptoms observed 1-3 weeks after transplanting.
- Inward leaf rolling, drooping, yellowing and withering of tillers.
- Lesions turn yellow to white as the disease advances
- Severely infected leaves tend to dry quickly
- Entire plant wilt completely
- If seedlings or young pants are infected they show wilting a stage known as Kresek stage.
- Affected tillers can be confusion with **stem borer** affected tillers which are easily pulled out while that of Kresek not.

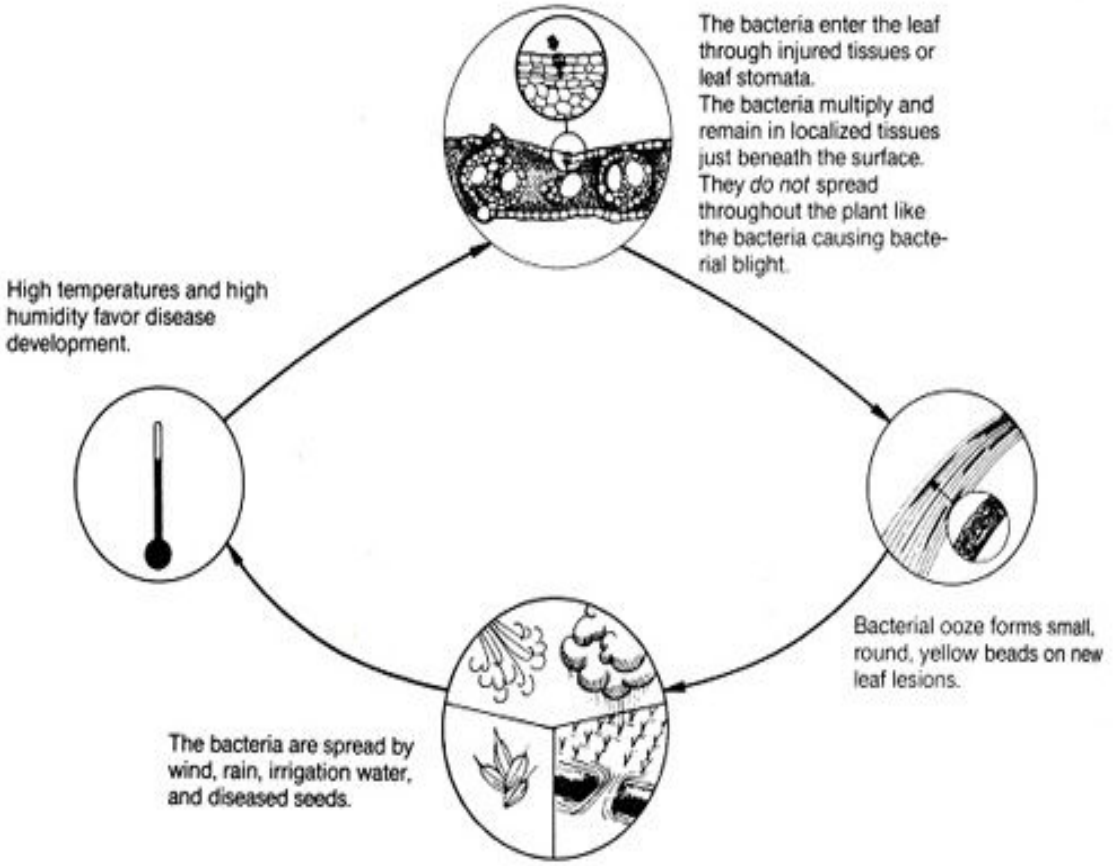
Leaf blight:

- It is most commonly seen.
- Blight phage of the disease usually appears 4-6 weeks after transplanting.
- Water-soaked to yellowish stripes on leaf blades or starting at leaf tips then later increase in length and width with a wavy margin
- Yellow leaf or pale yellow of mature plants
- Green water-soaked layer along the cut portion or leaf tip of leaves as early symptom

- Lesions later become grayish from growth of various saprophytic fungi
- Youngest leaf is uniform pale yellow or has broad yellow stripe.
- Older leaves do not show symptoms.
- Appearance of bacterial ooze that looks like a milky or opaque dew drop on young lesions early in the morning.
- Panicles sterile and unfilled but not stunted under severe conditions
- Symptoms appear on the margins of leaf blade and sheath as small, linear, water-soaked areas that soon elongate and coalesce into irregular, narrow, yellowish and brownish stripes.
- Droplets of white exudates are found on the stripes.
- During severe infection, the leaves become yellow and start dying from the tip downwards.



Causal organism: *X. campestris p.v oryzae* (Ishiyama) Dye. It is rod shaped bacterium with rounded ends, 1-2 X 0.8-1µm and monotrichus flagellum of 6-8 µm. it is gram negative and non spore forming.



Disease Management:

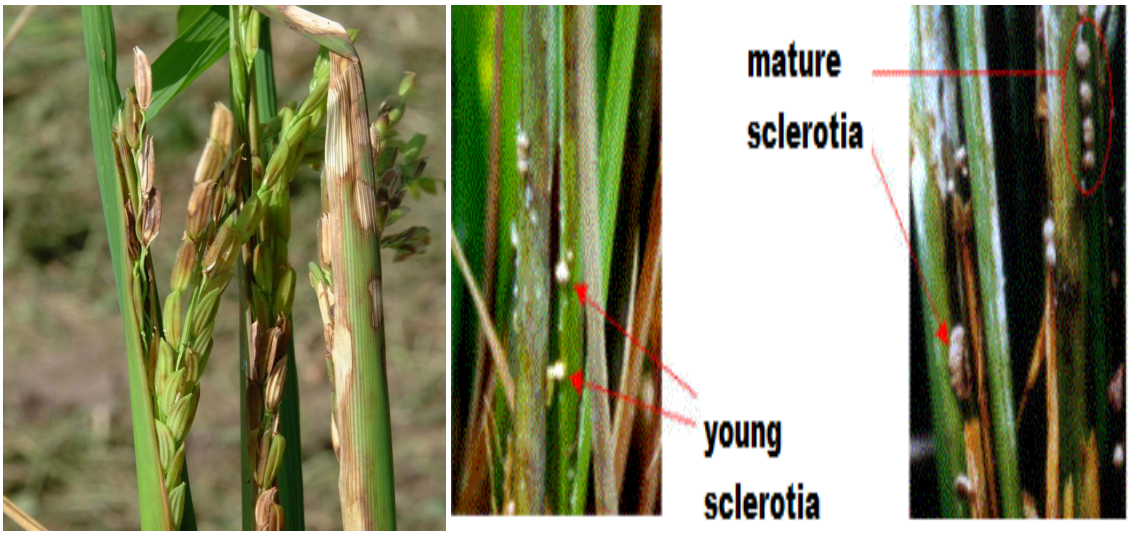
- Practicing field sanitation such as removing weed hosts, rice straws, rations and volunteer seedlings is important to avoid infection caused by this disease.
- Use of certified disease free seed and crop rotation.
- **Hot water treatment:** seed soaking for 12 hrs and then dipping in hot water at 53^0C for 30 minutes.
- Seed treatment with bleaching powder {Calcium oxychloride $Ca(Cl\ O)_2$} (100µg/ml) and zinc sulfate (2%) and application of stable bleaching powder @ 12.5 kg/ha can reduce the intensity of the disease.
- Seed soaking for 8 hr in Ceresan (0.1%) and steptacycline-crude agricultural preparation (3g in 1100 litre of water) is effective to eradicate seed infection.
- Foliar spray of Agrimycin-100 (250 ppm) or Copper fungicides alternatively with Streptocycline (250 ppm) or mixture of Agrimycin-100 and Fytolan (Copper Oxychloride) in the ratio of 50:500 ppm can significantly reduce the disease in field.
- Grow resistant variety like TKM-6, Rudra, Pathara, Kesari, Annapurna, Daya, Gayatri , Suphala, IR- 36,42 IR 20, IR 64, Swarna, PR 113, PR 114, PR 115, PR 116, Saket-4, Pant Dhan-4, Pant Dhan-6, Pant Dhan-10 etc.
- Moderate level of 60-80 kg N/ha with required potassium may be recommended in endemic areas during the wet season. The nitrogen should be applied in 3-4 splits.

4. Sheath Blight:

Sheath blight is one of the serious diseases of rice and sometimes important to other cereals as well. It is found worldwide wherever rice is grown. The banded leaf blight symptoms of this disease have been reported from Uttar Pradesh.

Symptoms:

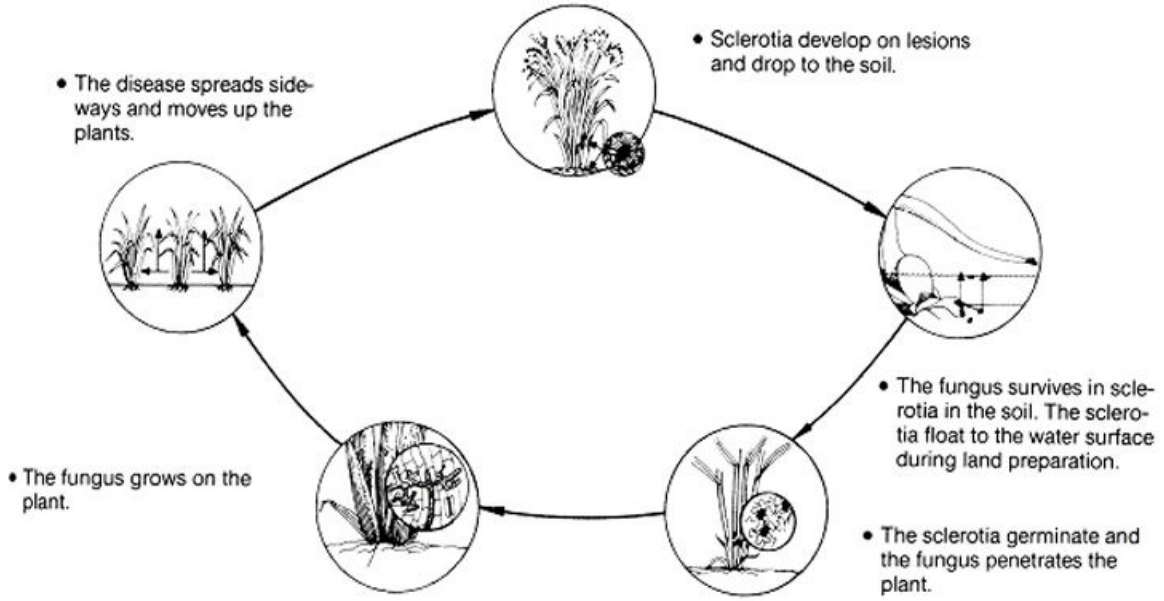
- Initial lesions are small, ellipsoidal or ovoid, greenish gray and water soaked and usually develop near the water line in lower leaves
- The symptoms develop as large, irregular, and oval to elliptical, green grey, water-soaked lesions on the sheath of leaves and have a straw coloured centre and a wide reddish brown margin.
- These lesions first develop near the water line on sheath or lower leaves when plants are in the late tillering or early internodes elongation stage.
- These lesions usually develop just below the leaf collar about 1/4 inch wide and 1/2 to 1.1/4 inch long. With age, the lesions expand and the centre of the lesions may become bleached with an irregular tan to brown border.
- Older lesions are elliptical or ovoid with a grayish white center and light brown to dark brown margin. Lesions may coalesce forming bigger lesions with irregular outline and may cause the death of the whole leaf.
- Seedlings and mature plants are blighted when humidity exceeds 95% and temperature in the range of 15-35 ^{0}C.
- Disease development progresses very rapidly in the early heading and grain filling growth stages during period of frequent rainfall and overcast sky.
- The grains in the lower parts of panicle are poorly filled.
- Additional losses result from increased lodging or reduced ratoon production due to reduced carbohydrate reserves.
- Near maturity of the plants dark brown coloured sclerotia are produced superficially on or near the lesions and easily fell down on soil.



Sheath blight of rice

Pathogen: ***Rhizoctonia solani (Thanatephorus cucumeris* - Perfect state)** other perfect stages is *Hypochnus sasakii* Shirai = *Corticium sasakii* (Shirai) Matsumoto = *Thanatephorus cucumeris* (Frank) Dark.

Disease Cycle: The **primary source of inoculum comes from sclerotia** and they survive between the crops. When the rice crop is planted, the sclerotia float on the surface of flooded water and infect the plants near the water line. Sclerotia can survive one to several years in soil. Further spread takes place by rain, irrigation and tools carrying soil contaminated with the pathogen. The infection can take place with a temperature ranging from 15 to35 C and 95% humidity. The pathogen can infect and survive on certain weed hosts which may also serve as the source of inoculum for further spread of the disease. The mycelium grows inside the tissues in all directions, initiating secondary spots, in turn producing sclerotia on the spots. The disease is highly destructive during humid and warm temperatures. High dose of nitrogenous fertilizers make the tissue more susceptible to the disease while high potassium induces resistance to the disease.



Disease Management:

- General sanitation-destruction of crop debris, crop rotation, mulching during summer may be helpful.
- Flooding of rice fields.
- Proper spacing.
- Efficient use of N-fertilizers.
- New vaieties and changing cultural practices often combine many of the factors that favour disease development. In recent years, wide acceptance of susceptible varieties, because of their high yielding potential, has contributed greatly to the rapid increase in the incidence of sheath blight.
- To manage the disease, application of heavy doses of nitrogen should be avoided as it predisposes plants to infection.
- Close transplanting of rice plants and wet and poorly drained fields for cultivation of rice should be avoided.
- The fields may be kept clean with grasses and weeds.
- Resistant varieties if available should be used and foliar fungicides may be economical for reducing sheath blight infection.
- In heavily infected patches, soil drench with 0.1% wet Ceresan will be useful.
- Foliar spray of validamycin (Rhizocin 3 L or Sheathmar @ 2.5 ml/liter water) or hexaconazole (Contaf or Sitara 5 EC @ 2 ml/liter water), Carbendazim 0.1 per cent, Propineb 0.1 per cent and Propiconazole 0.1 per cent or Tilt (Propioconazole), a combination of fungicides (Azoxystrobin 18.2% + Difenoconazole 11.4% SC) was found highly effective against rice sheath blight.
- Biocontrol : **Trichoderma viride, Pseudomonas fluorescens.**

5. Khaira disease

Economic Importance: Dr. Y.L.Nene and his associates first discovered khaira disease of rice in the region of the U.P. now Uttranchal. Khaira disease of rice is an excellent example of a disease due to non availability of Zinc to the plants in an othervise normal soil.

Symptom

- Usually in **nursery;** chlorotic / yellow patches at leaf base on both sides of the midrib; restricted root growth and usually main roots turn brown.
- Khaira disease usually appears when rice plants are in nursery.
- It appears in patches, after 10-15 days of transplanting.
- The diseased plant remains stunted.
- In the young leaves interveinal chlorosis can be seen.
- On **older leaves**, a large number of small, brown to bronze spots appear, which later coalesce and form bigger spots.
- Finally, entire leaf turns bronze coloured and dries up.
- The main roots turn brown and root growth is also restricted.
- In severe cases, plants fail to grow further and produce no ears.


Management

- This disease occurs due to deficiency of zinc, it can be corrected after observing first sign of deficiency.
- Use 25 kg $Znso_4$/ha before transplanting or sowing at time of land preparation.
- Spraying of a mixture of 5 kg of zinc sulphate and 2.5 kg of lime in 1000 litres of water after 10 days of sowing in the nursery is recommended.
- Second spray should be given after 20 days of sowing in the nursery again.
- The third and last spray is to be given in the field after 15-20 days of transplanting when its symptoms appear in the field.
- The zinc sulphate dust can also be applied @ 25 -30 kg/ ha before sowing if preceding crop has shown the symptoms of zinc deficiency.

6. Tungro Disease of Rice

Tungro disease is most destructive to rice in countries of Southeast Asia. Outbreaks of the disease are common in one part or the other in India. In severe cases of infection or infection during early stages of plant growth the yield loss may be as high as 100%. The damage depends on the variety used, plant stage at the time of infection, strain of the causal virus and environmental conditions.

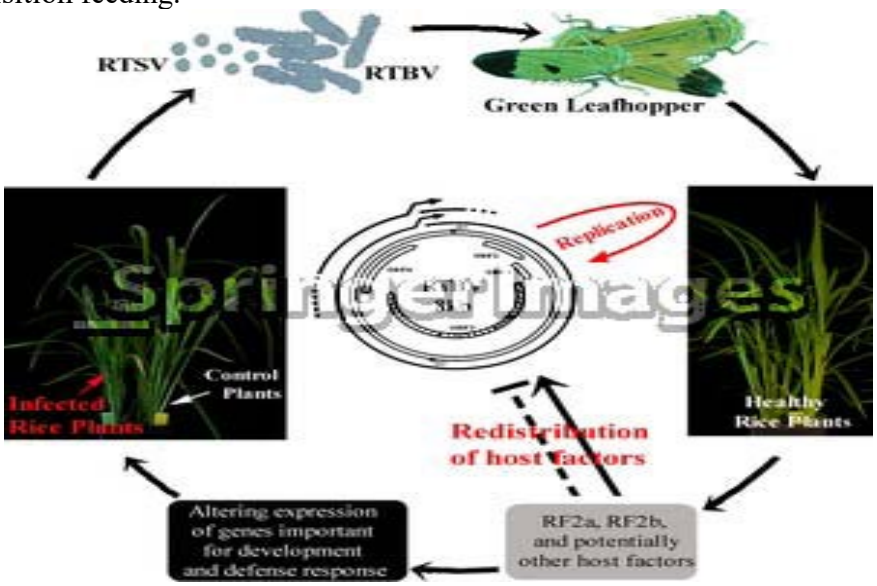
Symptoms:

- Affected plants show discoloration of the leaves which begins at the leaf tip and move down to the lower leaf portion.
- Young infected leaves show interveinal chlorosis and incomplete emergence.
- The tillering is reduced and plants become stunted.
- The flowering on infected plants may be delayed and most panicles are sterile or with partially filled grains with dark brown spots.
- The symptoms may be confused with nitrogen and zinc deficiencies or water stress or insect damage or other virus diseases like grassy stunt and orange leaf.


Pathogen: Tungro disease is associated with rice tungro bacilliform virus (RTBV) and rice tungro spherical virus (RTSV). Both the viruses appear responsible to cause infection simultaneously by the leafhopper, *Nephotettix virescens* (Distant). RTBV can not be transmitted by leafhopper vector in the absence of RTSV. RTBV particles are bscilliform in shape measuring 100 to 130 nm in length and 30 to 35 nm in width and contain ds DNA of 8.3 kb. RTSV particles are isometric and 30nm in diameter and contain ssRNA of about 12kb.

Disease cycle:

- The inoculum of the virus survives on rice or some wild relatives in the nature from where the infection takes place to the rice crop plants by the leafhopper vector.
- The vector can acquire the virus while feeding on infected plants within a feeding period of minimum 30 minutes and transmit it immediately when feed to healthy plants for a few minutes.
- After acquisition of the virus the leafhopper can transmit the virus from 5 to 8 days and after that become non-viruliferous or no virus is retained by the vector and would require reacquisition feeding.



Disease Management:

- Planting of resistant varieties either to vector or virus is the most economical means of managing the disease.
- **Eradication of tungro hosts-destroy stubbles.**
- Eliminating the hosts of the viruses and vector and to destroy stubbles after harvest is also advisable.
- **The use of resistant varieties- IR 36, IR 50, ADT 37, Ponmani, Co 45, Co 48, Surekha, Vikramarya, Bharani, IR 36 and white ponni .**
- **Elimination of vectors:-- light traps**
- **Spraying Monocrotophos @ 1.5 to 2.0 ml/litre of water** or Phosphamidan @1ml/lt or Ethophenphos@1.5ml/lt or Neem oil @3 per cent in the main field 15 and 30 days after transplanting to control leaf hoppers
- **Crop rotation.**